=> index bioscience medicine

INDEX 'ADISCTI, ADISINSIGHT, ADISNEWS, AGRICOLA, ANABSTR, ANTE, AQUALINE, AQUASCI, BIOENG, BIOSIS, BIOTECHABS, BIOTECHDS, BIOTECHNO, CABA, CAPLUS, CEABA-VTB, CIN, CONFSCI, CROPB, CROPU, DDFB, DDFU, DGENE, DISSABS, DRUGB, DRUGMONOG2, DRUGU, EMBAL, EMBASE, ...' ENTERED AT 13:46:40 ON 21 MAR 2007

71 FILES IN THE FILE LIST IN STNINDEX

=> S ((amylase (w) binding (w) protein)or (amylase with interacting) or amylase-binding)
1 FILE AGRICOLA
1 FILE ANABSTR
1 FILE BIOENG
51 FILE BIOSIS
3 FILE BIOTECHABS
3 FILE BIOTECHDS
18 FILE BIOTECHNO
13 FILES SEARCHED...
1 FILE CABA
91 FILE CAPLUS
3 FILE CONFSCI
1 FILE DDFB
8 FILE DGENE
23 FILES SEARCHED...
5 FILE DISSABS
1 FILE DRUGB
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244 FILE GENBANK
6 FILE JICST-EPLUS
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48 FILES SEARCHED...
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9 FILE TOXCENTER
14 FILE USPATFULL
2 FILE WPIDS
2 FILE WPINDEX
68 FILES SEARCHED...

27 FILES HAVE ONE OR MORE ANSWERS, 71 FILES SEARCHED IN STNINDEX

L1 QUE ((AMYLASE (W) BINDING (W) PROTEIN) OR (AMYLASE WITH INTERACTING) OR AM YLASE-BINDING)

=> d rank
F1 244 GENBANK
F2 91 CAPLUS
F3 52 SCISEARCH
F4 51 BIOSIS
F5 37 MEDLINE
F6 26 EMBASE
F7 20 ESBIOBASE
F8 18 BIOTECHNO
F9 14 PASCAL
F10 14 USPATFULL
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F12 9 TOXCENTER
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F14 6 JICST-EPLUS
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F17 3 BIOTECHDS
F18 3 CONFSCI

F19 2 WPIDS
F20 2 WPINDEX
F21 1 AGRICOLA
F22 1 ANABSTR
F23 1 BIOENG
F24 1 CABA
F25 1 DDFB
F26 1 DRUGB
F27 1 FSTA

=> File f2-f12
COST IN U.S. DOLLARS SINCE FILE ENTRY TOTAL SESSION
FULL ESTIMATED COST 3.15 4.83

FILE 'CAPLUS' ENTERED AT 13:49:33 ON 21 MAR 2007
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FILE 'USPATFULL' ENTERED AT 13:49:33 ON 21 MAR 2007
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=> S L1
7 FILES SEARCHED...
L2 345 L1

=> S (caries or plaque or coloniz?)(s) L2
L3 56 (CARIES OR PLAQUE OR COLONIZ?)(S) L2

=> S (treat? or prevent? or control?) (s) L3
6 FILES SEARCHED...
8 FILES SEARCHED...
L4 1 (TREAT? OR PREVENT? OR CONTROL?) (S) L3

=> S (treat? or prevent? or control?) and L3
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L5 6 (TREAT? OR PREVENT? OR CONTROL?) AND L3

=> S (dental or oral) and L2

L6 126 (DENTAL OR ORAL) AND L2

=> S (caries or plaque or coloniz?)and L6
L7 81 (CARIES OR PLAQUE OR COLONIZ?) AND L6

=> S (treat? or prevent? or control?)and L7
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L8 14 (TREAT? OR PREVENT? OR CONTROL?) AND L7

=> dup rem L8
PROCESSING COMPLETED FOR L8
L9 12 DUP REM L8 (2 DUPLICATES REMOVED)

=> d ibib abs L9 1-12

L9 ANSWER 1 OF 12 USPATFULL on STN
ACCESSION NUMBER: 2006:143506 USPATFULL <<LOGINID::20070321>>
TITLE: Compositions for ***treating*** cystic fibrosis
INVENTOR(S): Budny, John A., Westlake Village, CA, UNITED STATES
Budny, Matthew J., Westlake Village, CA, UNITED STATES

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2006121019	A1	20060608
APPLICATION INFO.:	US 2005-242447	A1	20051003 (11)

RELATED APPLN. INFO.: Continuation of Ser. No. US 2001-875997, filed on 6 Jun 2001, ABANDONED Continuation-in-part of Ser. No. US 2000-587818, filed on 6 Jun 2000, GRANTED, Pat. No. US 6830745 Continuation-in-part of Ser. No. US 1999-249674, filed on 12 Feb 1999, GRANTED, Pat. No. US 6159447 Continuation-in-part of Ser. No. US 1997-951393, filed on 16 Oct 1997, GRANTED, Pat. No. US 5871714
DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: COLIN P ABRAHAMS, 5850 CANOGA AVENUE, SUITE 400, WOODLAND HILLS, CA, 91367, US
NUMBER OF CLAIMS: 29
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 2 Drawing Page(s)
LINE COUNT: 693
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB A composition for degrading biofilm structure associated with cystic fibrosis and the debris associated therewith comprises an enzyme selected for its ability to dismantle the biofilm structure, and an anchor molecule coupled to an enzyme to form an enzyme-anchor complex. The anchor molecule is selected for its ability to attach to a surface on or proximal the biofilm structure. The attachment to the surface permits prolonged retention time of the enzyme-anchor complex where the biofilm structure and associated debris are present.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L9 ANSWER 2 OF 12 USPATFULL on STN
ACCESSION NUMBER: 2005:239995 USPATFULL <<LOGINID::20070321>>
TITLE: Methods and compositions for promoting ***oral*** health, and polypeptides useful for same
INVENTOR(S): Gregory, Richard L., Carmel, IN, UNITED STATES
Catt, Diana M., Mooresville, IN, UNITED STATES
PATENT ASSIGNEE(S): Advanced Research and Technology Institute, Inc. (U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2005207995	A1	20050922
APPLICATION INFO.:	US 2004-828837	A1	20040421 (10)

RELATED APPLN. INFO.: Continuation-in-part of Ser. No. US 9004, ABANDONED A 371 of International Ser. No. WO 2000-US11992, filed on 3 May 2000

NUMBER DATE
---------- --------
PRIORITY INFORMATION: US 1999-132312P 19990503 (60)
DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: Woodard, Emhardt, Moriarty, McNett & Henry LLP, Bank One Center/Tower, Suite 3700, 111 Monument Circle, Indianapolis, IN, 46204-5137, US
NUMBER OF CLAIMS: 42
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 16 Drawing Page(s)
LINE COUNT: 2145
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB Described are methods and compositions for promoting ***oral*** health in a host that involve the administration of a 65 kDa protein from S. mutans or a fragment or functional analog thereof, to the ***oral*** cavity of the host. The 65 kDa protein exhibits murein hydrolase enzyme activity and in inventive embodiments the enzyme or active fragments can be used to lyse bacteria commonly present in ***dental*** ***plaque*** or other surfaces of the ***oral*** cavity. Methods for ***controlling*** ***dental*** ***caries*** include administering non-immunogenic polypeptides from bacterial adhesions to competitively block bacterial attachment without implicating the immune system of the host. Novel isolated DNA encoding the 65 kDa protein or a fragment thereof, as well as recombinant forms of the protein or fragment, and methods for their production, are also described.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L9 ANSWER 3 OF 12 EMBASE COPYRIGHT (c) 2007 Elsevier B.V. All rights reserved on STN DUPLICATE 1
ACCESSION NUMBER: 2003384895 EMBASE <<LOGINID::20070321>>
TITLE: ***Amylase*** - ***binding*** proteins A (AbpA) and B (AbpB) differentially affect ***colonization*** of rats' teeth by Streptococcus gordonii.
AUTHOR: Tanzer J.M.; Grant L.; Thompson A.; Li L.; Rogers J.D.; Haase E.M.; Scannapieco F.A.
CORPORATE SOURCE: J.M. Tanzer, School of Dental Medicine, Univ. of Connecticut Health Center, Farmington, CT 06030-1605, United States. tanzer@nso.uchc.edu
SOURCE: Microbiology, (1 Sep 2003) Vol. 149, No. 9, pp. 2653-2660.

Refs: 35
ISSN: 1350-0872 CODEN: MROBEO
COUNTRY: United Kingdom
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: 004 Microbiology
005 General Pathology and Pathological Anatomy
011 Otorhinolaryngology
LANGUAGE: English
SUMMARY LANGUAGE: English
ENTRY DATE: Entered STN: 9 Oct 2003
Last Updated on STN: 9 Oct 2003
AB Streptococcus gordonii produces two .alpha.- ***amylase*** - ***binding*** proteins, AbpA and AbpB, that have been extensively studied in vitro. Little is known, however, about their significance in ***oral*** ***colonization*** and cariogenicity (virulence). To clarify these issues, weanling specific pathogen-free Osborne-Mendel rats, TAN : SPFOM(OM)BR, were inoculated either with wild-type strains FAS4-S or Challis-S or with strains having isogenic mutations of abpA, abpB, or both, to compare their ***colonization*** abilities and persistence on the teeth. Experiments were done with rats fed a sucrose-rich diet containing low amounts of starch or containing only starch. The mutants and wild-types were quantified in vivo and carious lesions were scored. In 11 experiments, S. gordonii was a prolific ***colonizer*** of the teeth when rats were fed the sucrose (with low starch)-supplemented diet, often dominating the flora. Sucrose-fed rats had several-fold higher recoveries of inoculants than those eating the sucrose-free, starch-supplemented diet, regardless of inoculant type. The strain

defective in AbpB could not ***colonize*** teeth of starch-only-eating rats, but could ***colonize*** rats if sucrose was added to the diet. Strains defective in AbpA surprisingly ***colonized*** better than their wild-types. A double mutant deficient in both AbpA and AbpB (abpA/abpB) ***colonized*** like its wild-type. Wild-types FAS4-S and Challis-S had no more than marginal cariogenicity. Notably, in the absence of AbpA, cariogenicity was slightly augmented. Both the rescue of ***colonization*** by the AbpB(-) mutant and the augmentation of ***colonization*** by AbpA(-) mutant in the presence of dietary sucrose suggested additional ***amylase*** - ***binding*** ***protein*** interactions relevant to ***colonization*** . Glucosyltransferase activity was greater in mutants defective in abpA and modestly increased in the abpB mutant. It was concluded that AbpB is required for ***colonization*** of teeth of starch-eating rats and its deletion is partially masked if rats eat a sucrose-starch diet. AbpA appears to inhibit ***colonization*** of the ***plaque*** biofilm in vivo. This unexpected effect in vivo may be associated with interaction of AbpA with glucosyltransferase or with other ***colonization*** factors of these cells. These data illustrate that the complex nature of the ***oral*** environment may not be adequately modelled by in vitro systems.

L9 ANSWER 4 OF 12 SCISEARCH COPYRIGHT (c) 2007 The Thomson Corporation on STN
ACCESSION NUMBER: 2003:940134 SCISEARCH <<LOGINID::20070321>>
THE GENUINE ARTICLE: 736MN
TITLE: Bacterial ***colonization*** during de novo ***plaque*** formation
AUTHOR: Ramberg P (Reprint); Sekino S; Uzel N G; Socransky S; Lindhe J
CORPORATE SOURCE: Univ Gothenburg, Inst Odontol, Dept Periodontol, Box 450, SE-40530 Gothenburg, Sweden (Reprint); Univ Gothenburg, Inst Odontol, Dept Periodontol, SE-40530 Gothenburg, Sweden; Forsyth Inst, Dept Periodontol, Boston, MA USA
COUNTRY OF AUTHOR: Sweden; USA
SOURCE: JOURNAL OF CLINICAL PERIODONTOLOGY, (NOV 2003) Vol. 30, No. 11, pp. 990-995.
ISSN: 0303-6979.
PUBLISHER: BLACKWELL MUNKSGAARD, 35 NORRE SOGADE, PO BOX 2148, DK-1016 COPENHAGEN, DENMARK.
DOCUMENT TYPE: Article; Journal
LANGUAGE: English
REFERENCE COUNT: 45
ENTRY DATE: Entered STN: 7 Nov 2003
Last Updated on STN: 7 Nov 2003
ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS

AB Objective: To determine microbial changes that occur during ***plaque*** formation in a dentition free of gingival inflammation.

Material and Methods: Ten subjects were recruited. The study included one preparatory period (2 weeks) and a ***plaque*** accumulation period (4 days). The volunteers exercised proper tooth cleaning methods, were scaled and received repeated professional mechanical tooth cleaning during the preparatory period. During the ***plaque*** accumulation period, the participants abstained from ***plaque*** ***control*** measures. ***Plaque*** was scored on the approximal surfaces of maxillary and mandibular premolars on Days 0, 1, 2 and 4 using a scale from 0 to 5 and according to the criteria of the Quigley and Hein ***Plaque*** Index (QHI). Supragingival ***plaque*** samples were obtained from the same intervals and surfaces and evaluated using a checkerboard DNA-DNA hybridization technique.

Results: The mean QHI increased from 0 to 1.6 (Day 4). The total number of organisms on Day 0 averaged 140x10(5) and increased to about 210x10(5) after 4 days without ***oral*** hygiene.

The most dominant species on Day 0 were members of the genus Actinomyces. These organisms comprised almost 50% of the microbiota evaluated. None of the Actinomyces species increased significantly during the 4 days. Some Streptococcus species increased significantly over time as well as species of the genera Capnocytophaga, Campylobacter, Fusobacteria and Actinomyces actinomycetemcomitans.

Conclusion: In the present investigation, the preparatory phase

established a situation with minimal gingival inflammation and close to zero amounts of ***dental*** ***plaque*** . The Day 0 ***plaque*** samples exhibited high proportions of Actinomyces species. During the 4 days of no ***oral*** hygiene, there was a small increase in total numbers of organisms as well as a modest increase in the proportion of "disease-associated" taxa such as species of the "orange complex" species.

L9 ANSWER 5 OF 12 USPATFULL on STN
ACCESSION NUMBER: 2002:66609 USPATFULL <<LOGINID::20070321>>
TITLE: Compositions for ***treating*** biofilm
INVENTOR(S): Budny, John A., Westlake Village, CA, UNITED STATES
Budny, Matthew J., Westlake Village, CA, UNITED STATES

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2002037260	A1	20020328
APPLICATION INFO.:	US 2001-876248	A1	20010606 (9)

RELATED APPLN. INFO.: Continuation-in-part of Ser. No. US 2000-587818, filed on 6 Jun 2000, PENDING Continuation-in-part of Ser. No. US 1999-249674, filed on 12 Feb 1999, GRANTED, Pat. No. US 6159447 Continuation-in-part of Ser. No. US 1997-951393, filed on 16 Oct 1997, GRANTED, Pat. No. US 5871714
DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: COLIN P ABRAHAMS, 5850 CANOGA AVENUE, SUITE 400, WOODLAND HILLS, CA, 91367
NUMBER OF CLAIMS: 35
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 1 Drawing Page(s)
LINE COUNT: 1056
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB A composition for ***treating*** a biofilm comprises a first anchor enzyme component to degrade biofilm structures and a second anchor enzyme component having the capability to act directly upon the bacteria for a bactericidal effect.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L9 ANSWER 6 OF 12 USPATFULL on STN
ACCESSION NUMBER: 2002:37289 USPATFULL <<LOGINID::20070321>>
TITLE: Compositions for ***treating*** cystic fibrosis
INVENTOR(S): Budny, John A., Westlake Village, CA, UNITED STATES
Budny, Matthew J., Westlake Village, CA, UNITED STATES

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2002022005	A1	20020221
APPLICATION INFO.:	US 2001-875997	A1	20010606 (9)

RELATED APPLN. INFO.: Continuation-in-part of Ser. No. US 2000-587818, filed on 6 Jun 2000, PENDING Continuation-in-part of Ser. No. US 1999-249674, filed on 12 Feb 1999, GRANTED, Pat. No. US 6159447 Continuation-in-part of Ser. No. US 1997-951393, filed on 16 Oct 1997, GRANTED, Pat. No. US 5871714
DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: COLIN P ABRAHAMS, 5850 CANOGA AVENUE, SUITE 400, WOODLAND HILLS, CA, 91367
NUMBER OF CLAIMS: 29
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 1 Drawing Page(s)
LINE COUNT: 761
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB A composition for degrading biofilm structure associated with cystic fibrosis and the debris associated therewith comprises an enzyme selected for its ability to dismantle the biofilm structure, and an anchor molecule coupled to an enzyme to form an enzyme-anchor complex. The anchor molecule is selected for its ability to attach to a surface

on or proximal the biofilm structure. The attachment to the surface permits prolonged retention time of the enzyme-anchor complex where the biofilm structure and associated debris are present.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L9 ANSWER 7 OF 12 EMBASE COPYRIGHT (c) 2007 Elsevier B.V. All rights reserved on STN DUPLICATE 2
ACCESSION NUMBER: 2001376478 EMBASE <<LOGINID::20070321>>
TITLE: Role of Streptococcus gordonii ***amylase*** - ***binding*** ***protein*** A in adhesion to hydroxyapatite, starch metabolism, and biofilm formation.
AUTHOR: Rogers J.D.; Palmer R.J. Jr.; Kolenbrander P.E.; Scannapieco F.A.
CORPORATE SOURCE: F.A. Scannapieco, Department of Oral Biology, School of Dental Medicine, State University of New York, Buffalo, NY 14214, United States. fasl@acsu.buffalo.edu
SOURCE: Infection and Immunity, (2001) Vol. 69, No. 11, pp. 7046-7056.
Refs: 52
ISSN: 0019-9567 CODEN: INFIBR
COUNTRY: United States
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: 004 Microbiology
011 Otorhinolaryngology
LANGUAGE: English
SUMMARY LANGUAGE: English
ENTRY DATE: Entered STN: 15 Nov 2001
Last Updated on STN: 15 Nov 2001
AB Interactions between bacteria and salivary components are thought to be important in the establishment and ecology of the ***oral*** microflora. alpha.-Amylase, the predominant salivary enzyme in humans, binds to Streptococcus gordonii, a primary ***colonizer*** of the tooth. Previous studies have implicated this interaction in adhesion of the bacteria to salivary pellicles, catabolism of dietary starches, and biofilm formation. ***Amylase*** ***binding*** is mediated at least in part by the ***amylase*** - ***binding*** ***protein*** A (AbpA). To study the function of this protein, an erythromycin resistance determinant [erm(AM)] was inserted within the abpA gene of S. gordonii strains Challis and FAS4 by allelic exchange, resulting in abpA mutant strains Challis-E1 and FAS4-E1. Comparison of the wild-type and mutant strains did not reveal any significant differences in colony morphology, biochemical metabolic profiles, growth in complex or defined media, surface hydrophobicity, or coaggregation properties. Scatchard analysis of adhesion isotherms demonstrated that the wild-type strains adhered better to human parotid-saliva- and amylase-coated hydroxyapatite than did the AbpA mutants. In contrast, the mutant strains bound to whole-saliva-coated hydroxyapatite to a greater extent than did the wild-type strains. While the wild-type strains preincubated with purified salivary amylase grew well in defined medium with potato starch as the sole carbohydrate source, the AbpA mutants did not grow under the same conditions even after preincubation with amylase. In addition, the wild-type strain produced large microcolonies in a flow cell biofilm model, while the abpA mutant strains grew much more poorly and produced relatively small microcolonies. Taken together, these results suggest that AbpA of S. gordonii functions as an adhesin to amylase-coated hydroxyapatite, in salivary-amylase-mediated catabolism of dietary starches and in human saliva-supported biofilm formation by S. gordonii.

L9 ANSWER 8 OF 12 CAPLUS COPYRIGHT 2007 ACS on STN
ACCESSION NUMBER: 2000:790533 CAPLUS <<LOGINID::20070321>>
DOCUMENT NUMBER: 133:355015
TITLE: Streptococcus mutans fimbrial adhesin compositions and recombinant SmaA polypeptides for ***controlling*** ***dental*** ***caries***
INVENTOR(S): Gregory, Richard L.
PATENT ASSIGNEE(S): Advanced Research & Technology Institute, Inc., USA
SOURCE: PCT Int. Appl., 67 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent

LANGUAGE: English
FAMILY ACC. NUM. COUNT: 2
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2000066616	A1	20001109	WO 2000-US11992	20000503

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CR, CU, CZ, DE, DK, DM, DZ, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW
RW: GH, GM, KE, LS, MW, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG

US 2005207995	A1	20050922	US 2004-828837	20040421

PRIORITY APPLN. INFO.: US 1999-132312P P 19990503
WO 2000-US11992 W 20000503
US 2001-9004 B2 20011105

AB Described are methods and compns. for ***controlling*** ***dental*** ***caries*** in a host which involve the administration of a fimbrial-assocd. adhesin from S. mutans , SmaA, or a fragment thereof, to the ***oral*** cavity of the host. Methods for ***controlling*** ***dental*** ***caries*** include administering non-immunogenic polypeptides from bacterial adhesins to competitively block bacterial attachment without implicating the immune system of the host. Novel isolated DNA encoding SmaA or a fragment thereof, as well as recombinant SmaA protein of fragment, and methods for their prodn., are also described.

REFERENCE COUNT: 1 THERE ARE 1 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L9 ANSWER 9 OF 12 SCISEARCH COPYRIGHT (c) 2007 The Thomson Corporation on STN
ACCESSION NUMBER: 2000:59428 SCISEARCH <<LOGINID::20070321>>
THE GENUINE ARTICLE: 274EF
TITLE: Adhesion of Candida albicans to ***oral*** streptococci is promoted by selective adsorption of salivary proteins to the streptococcal cell surface
AUTHOR: O'Sullivan J M (Reprint); Jenkinson H F; Cannon R D
CORPORATE SOURCE: Univ Otago, Dept Oral Sci & Orthodont, POB 647, Dunedin, New Zealand (Reprint); Univ Otago, Dept Oral Sci & Orthodont, Dunedin, New Zealand
COUNTRY OF AUTHOR: New Zealand
SOURCE: MICROBIOLOGY-SGM, (JAN 2000) Vol. 146, Part 1, pp. 41-48. ISSN: 1350-0872.
PUBLISHER: SOC GENERAL MICROBIOLOGY, MARLBOROUGH HOUSE, BASINGSTOKE RD, SPENCERS WOODS, READING RG7 1AE, BERKS, ENGLAND.
DOCUMENT TYPE: Article; Journal
LANGUAGE: English
REFERENCE COUNT: 48
ENTRY DATE: Entered STN: 2000
Last Updated on STN: 2000
ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS

AB Adhesion of Candida albicans to saliva-coated surfaces is an important early step in the ***colonization*** of the ***oral*** cavity, C. albicans cells also adhere to several species of ***oral*** streptococci including Streptococcus gordonii. Streptococcus oralis and Streptococcus sanguinis in what are believed to be multi-modal interactions. It is now demonstrated that incubation of streptococcal cells of these species with human parotid saliva further promotes the adhesion of C. albicans cells by up to 2.3-fold. Various species of streptococci were shown to adsorb different protein components of parotid saliva to their cell surfaces, The basic proline-rich proteins (bPRPs), to which C. albicans cells bind on nitrocellulose blot overlay, were strongly adsorbed to the surface of S. gordonii cells but not to S. oralis cells. Parotid saliva that was pre-adsorbed with S. gordonii cells and then applied to hydroxylapatite beads was <50 % effective at supporting adhesion of C, albicans compared with ***control*** (non-adsorbedd) saliva, demonstrating that bPRPs are major pellicle receptors, C. albicans

cells did not adsorb bPRPs from fluid-phase parotid saliva. Following size-exclusion chromatography of parotid saliva samples. pooled fractions enriched in bPRPs promoted maximal adhesion of C. albicans to 5, gordonii cells. The results demonstrate that C. albicans cells recognize only surface-bound forms of bPRPs and suggest that these proteins adsorbed to enamel or to streptococcal surfaces promote C. albicans adhesion and ***oral*** ***colonization*** .

L9 ANSWER 10 OF 12 SCISEARCH COPYRIGHT (c) 2007 The Thomson Corporation on STN
ACCESSION NUMBER: 1995:720538 SCISEARCH <<LOGINID::20070321>>
THE GENUINE ARTICLE: TA263
TITLE: ***DENTAL*** ***PLAQUE*** AS A BIOFILM
AUTHOR: MARSH P D (Reprint); BRADSHAW D J
CORPORATE SOURCE: PUBL HLTH LAB SERV, CTR APPL MICROBIOL & RES, DIV MICROBIAL PATHOGEN, SALISBURY SP4 0JG, WILTS, ENGLAND (Reprint)
COUNTRY OF AUTHOR: ENGLAND
SOURCE: JOURNAL OF INDUSTRIAL MICROBIOLOGY, (SEP 1995) Vol. 15, No. 3, pp. 169-175.
ISSN: 0169-4146.
PUBLISHER: STOCKTON PRESS, HOUNDMILLS, BASINGSTOKE, HANTS, ENGLAND RG21 2XS.
DOCUMENT TYPE: Article; Journal
FILE SEGMENT: AGRI
LANGUAGE: English
REFERENCE COUNT: 62
ENTRY DATE: Entered STN: 1995
Last Updated on STN: 1995
ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS
AB ***Dental*** ***plaque*** is the diverse microbial community found on the tooth surface embedded in a matrix of polymers of bacterial and salivary origin, Once a tooth surface is cleaned, a conditioning film of proteins and glycoproteins is adsorbed rapidly to the tooth surface, ***Plaque*** formation involves the interaction between early bacterial colonisers and this film (the acquired enamel pellicle), To facilitate colonisation of the tooth surface, some receptors on salivary molecules are only exposed to bacteria once the molecule is adsorbed to a surface, Subsequently, secondary colonisers adhere to the already attached early colonisers (co-aggregation) through specific molecular interactions, These can involve protein-protein or carbohydrate-protein (lectin) interactions, and this process contributes to determining the pattern of bacterial succession. As the biofilm develops, gradients in biologically significant factors develop, and these permit the co-existence of species that would be incompatible with each other in a homogenous environment, ***Dental*** ***plaque*** develops naturally, but it is also associated with two of the most prevalent diseases affecting industrialised societies (***caries*** and periodontal diseases), Future strategies to ***control*** ***dental*** ***plaque*** will be targeted to interfering with the formation, structure and pattern of development of this biofilm.

L9 ANSWER 11 OF 12 SCISEARCH COPYRIGHT (c) 2007 The Thomson Corporation on STN
ACCESSION NUMBER: 1994:792382 SCISEARCH <<LOGINID::20070321>>
THE GENUINE ARTICLE: PW836
TITLE: COMPARISON OF ***AMYLASE*** - ***BINDING*** PROTEINS IN ***ORAL*** STREPTOCOCCI
AUTHOR: GWYNN J P (Reprint); DOUGLAS C W I
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ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS

AB Certain species of ***oral*** streptococci bind salivary amylase to their cell surface. The patterns of ***amylase*** - ***binding*** proteins produced by a range of streptococci have been compared by ligand blotting and several characteristics of the binding proteins investigated. Streptococcus gordonii was the most homogeneous species and almost all strains produced proteins migrating with molecular mass 82 kDa and 20 kDa. Other species were more heterogeneous, releasing proteins that resolved at 87 or 82 kDa and/or between 20 and 36 kDa. Binding of amylase to the 82/87-kDa proteins on ligand blots was ***prevented*** by amylase inhibitors, amylase substrates and periodate ***treatment*** but these had limited or no effect on ***amylase*** ***binding*** to 20-36 kDa proteins. Also, the 20 kDa protein of S. gordonii Challis was released into culture medium before the 82-kDa protein. These data suggest that there is significant variation in ***amylase*** - ***binding*** proteins among streptococci and that the high and low molecular mass proteins differ in the way they interact with salivary amylase.

L9 ANSWER 12 OF 12 BIOSIS COPYRIGHT (c) 2007 The Thomson Corporation on STN
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AB The purpose of the work described here was to investigate the mechanisms involved in the binding of salivary .alpha.-amylase to Streptococcus gordonii NCTC 7868 (Challis). Of six types of .alpha.-amylase studied, only mammalian forms of the enzyme were found to bind to S. gordonii cells. Salivary .alpha.- ***amylase*** ***binding*** was inhibited by ***treatment*** of cells with trypsin and pronase, but not with pepsin or sodium periodate. Presence of starch, dextrin, or maltoheptaose partially inhibited binding of the enzyme to S. gordonii. Both mutanolysin extracts of cells and culture supernatants contained .alpha.- ***amylase*** - ***binding*** activity, which was partially purified by Sepharose CL-6B and DEAE-ion-exchange chromatography. Western blotting detected four putative receptor bands-65 kDa, 12.5 kDa, and one with a very high molecular weight; the lower-molecular-weight components may be products of proteolytic degradation of the high-molecular-weight material, but their true relationship has yet to be determined. Pre- ***treatment*** of salivary .alpha.-amylase with these putative receptors partially inhibited subsequent binding of the enzyme to S. gordonii cells. When bound to cells, only 19% of the salivary .alpha.-amylase activity was detectable, suggesting that .alpha.-amylase binds to the receptor at or near the active site of the enzyme.

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L6 126 S (DENTAL OR ORAL) AND L2
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